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THE BLOOD FLOW THROUGH ACTIVE AND INACTIVE MUSCLES OF THE FOREARM DURING SUSTAINED HAND-GRIP CONTRACTIONS

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Despite the vasodilatation which occurs in a muscle during contraction, the full exploitation of this physiological response is hindered by the mechanical compression of the vessels by the contracting muscle (Gaskell, 1877), and its function is presumably thereby impaired. The continuous mechanical compression of the blood vessels through the active muscle has been generally accepted as the cause of the early onset of fatigue during sustained contractions; the validity of this view may be judged from the values of intramuscular pressure, of 150–300 mm Hg, determined during maximal isometric contractions of frog muscle (Hill, 1948) and of rabbit muscle (Mazella, 1954).

Grant (1938) and Barcroft & Dornhorst (1949) found small increases in the blood flowing through the muscles during both sustained and rhythmic contractions, while in a previous report from this laboratory Clarke, Hellon & Lind (1958) showed that at a tension of 1/3 maximal the increase of blood flow during contractions was greater as muscle temperature increased and could, in fact, be substantial. It is surprising, however, to find that there are no systematic measurements of the amount of blood flowing through muscles contracting at different tensions.

In addition to the direct measurements of blood flow mentioned above, some evidence has also been obtained by inference as to whether or not blood is actually flowing through the muscle vessels at different tensions. Dolgin & Lehmann (1930) found that the durations of weak contractions could be reduced by occluding the arterial inflow, but that in strong contractions this was not the case. Marschak (1931) claimed that the radial pulse could be obliterated during strong contractions of the forearm muscles. The implication from the results of these workers, that strong contractions were needed to occlude arterial inflow by means of intramuscular pressure, is in contrast to the findings of Barcroft & Millen (1939),

who studied calf muscles. These authors assessed the presence of blood flow by a method in which the temperature of 'pre-cooled' or 'pre-warmed' muscle was recorded with and without circulatory arrest, and deduced that the blood flow to the muscle was occluded at the surprisingly low tension of less than 20 % maximal. It seemed evident that the conflict apparent in the results, both direct and inferred, from the sources quoted above could best be resolved by a systematic examination of the amount of blood flowing through muscles contracting at different tensions.

The present experiments, then, were intended to determine this relationship, to estimate the tension at which intramuscular pressure overcomes the perfusion pressure of blood through the contracting muscle, and to assess the functional significance of the blood flow through the muscle during the contractions. Since the hand-grip contraction of the forearm involved some forearm muscles but not others, it was also necessary to determine to which of the muscles the blood flow was increased. Some of the results have been briefly reported (Humphreys & Lind, 1962*a*, *b*).

METHODS

Results were obtained from four subjects. As has been found before in this type of experiment (e.g. Lind, 1959), some training and a high degree of motivation were required of the subjects; training is necessary to keep involuntary movements of the forearm to a minimum to obtain good blood-flow records, motivation is necessary to persist with the contraction, despite associated pain towards the end, until the required tension could no longer be maintained.

Contractions were made on a simple hand-grip, strain-gauge dynamometer described earlier (Clarke *et al.* 1958) with the elbow held at a right angle. The maximal tension that could be exerted was determined at the start of each experiment by taking the average of the two greatest of three brief contractions (< 3 sec); 1 min intervals were allowed after each of these maximal contractions. In practice, these maxima did not vary by more than 2 %. The forearm was immersed in water for 30 min at a water temperature of 18, 26, 34 or 42° C after which a sustained contraction was made to the point of fatigue, at a submaximal tension of 30, 40, 50, 60 or 70 %. The end-point of each contraction was taken to be the moment when the subject could no longer maintain the required tension. The subject monitored his own performance by observing the deflexion of the spot on a galvanometer which resulted from the tension applied to the dynamometer. The duration of each contraction was noted to the nearest second.

Forearm blood flows were recorded on three subjects before, during and after contractions, by means of a Whitney (1953) strain-gauge plethysmograph. Contractions were performed on two separate occasions at each tension and muscle temperature, once with and once without blood-flow recordings, to ensure that the arterial cuff round the wrist did not affect the duration of the contraction. Before the contraction, blood flow was measured for 2 min. Recordings were continued throughout each contraction, when the flows were recorded four times/min, except in contractions which by experience were considered likely to be shorter than about 1·5 min, when they were recorded 5 times/min. The pressure in the venous collecting-cuff was 60 mm Hg; a further series of experiments were performed at 30 and 70 % of maximal tension with a venous collecting-cuff pressure of 90 mm Hg. There were no systematic differences in the results obtained at the two different collecting pressures. The

procedure adopted for analysis of the blood flows was that proposed by Cooper & Kerslake (1950).

Immediately the contractions were finished, recording of the blood flow through the forearm was continued for another 2 min. The arterial cuff round the wrist was then deflated and recording was discontinued for a short time. Measurements were again made of the blood flows for a further 2 min, 3·5 min after the contraction ended. This procedure was repeated, measuring the flows through the forearm for 2 min after 7 min had elapsed from the end of the contraction. In this way there were records of post-exercise blood flows during the periods 0–2, 3·5–5·5 and 7–9 min after the contraction had ended.

Blood flows recorded during sustained contractions could not always be measured because of involuntary movements of the arm. On such occasions the recording was rejected. At a tension of 70 % it was possible to interpret only five out of all the recordings made, and consequently these results have not been included in the graphs shown below.

An electromyographic study, bipolar needles being used, was made both to confirm the functional anatomy of the forearm muscles during the contractions, and to find regions of muscle for thermocouple needle insertion where pain, and muscle movement relative to the skin, were minimal. After completion of these preliminary electromyographic studies, thermocouples were inserted into the various muscles of the left arm, usually to a depth of 15–20 mm. At the most, four thermocouples were inserted at any one time. The arm was immersed in cool or in hot water and submaximal sustained contractions made as before. Muscle temperatures were recorded before, during and after the contractions. To confirm that the major changes in muscle temperature during the contractions were due to changes in blood flow, it was necessary to repeat the experiment with the blood flow to the forearm occluded by a sphygmanometer cuff round the upper arm at a pressure of 240 mm Hg. This technique differs only in detail from that described by Barcroft & Millen (1939).

RESULTS

Blood-flow measurements in the forearm

Forearm blood flows during sustained contractions at tensions from 30 to 60 % of maximal voluntary contraction (MVC), when the arm was immersed in water at 34° C, are shown in Fig. 1, where the increase of blood flow over the resting level is plotted against time for three subjects. Blood flow through the forearm was clearly present at all tensions up to 60 % MVC for all three subjects. At the start of each contraction there was usually an immediate and variable increase over the resting values, related, possibly, to the increase in blood pressure that was noted to occur as soon as the contractions had begun (see below). During each contraction there was a progressive increase in the amount of blood flowing through the forearm until fatigue occurred. At the lower tensions there was a steadily increasing rate of rise of blood flow, but in the stronger contractions the increase in blood flow appeared to be nearly linear. Flows in the early part of contractions showed fairly small increases over resting values (and therefore agree with the findings of Grant (1938)) but by the end of the contractions the levels of blood flow were quite high, and varied between 15 and 40 ml./100 ml./min greater than the resting levels, depending on the tension exerted.

The involuntary movements and the muscular tremor in the forearm were considerable at a tension of 70 % MVC so that, despite frequent attempts to measure blood flows in the forearms of all the subjects, only five individual recordings could be measured; the values of these were 1, 2, 2, 8 and 11 ml./100 ml./min increase over resting values. Thus, although it was clear that some increase in blood flow through the forearm could be detected even at this tension, the results were fragmentary and are not included in Fig. 1.

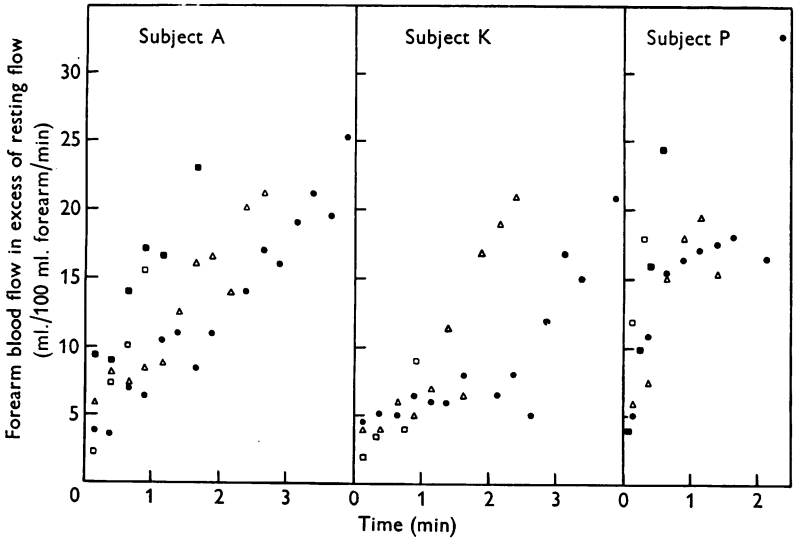

Fig. 1. Forearm blood flows, in excess of resting flows, for three subjects during contractions of 30 % (●), 40 % (△), 50 % (■) and 60 % (□) of maximal voluntary contraction. The forearms were immersed in a water-bath at 34° C.

Figure 2 shows the blood flows through the forearm for one subject for tensions from 30 to 60 % MVC when the arm was immersed in water at 18, 26 or 42° C. At each temperature, the changes in blood flow during the contractions followed the same pattern as that seen in Fig. 1, where water temperature was 34° C. The amount of blood flow through the forearm during the contractions increased with temperature at each tension. Clearly, from the results shown in Figs. 1 and 2, blood flow during sustained contractions increased substantially, even at high tensions when the intramuscular pressure may be presumed to have been considerable. In view of the important role imputed to the intramuscular pressure in restricting the blood flow in sustained contractions, it was desirable to obtain some estimate of its effect on the level of blood flow in contractions at different tensions. As is shown below (see Fig. 8), blood pressure increased during contractions and varied both with the tension exerted and

the length of time the tension was applied; an estimate of the effect of intramuscular pressure should presumably be made only when the blood pressure is comparable at different tensions—blood pressure appeared to be similar in the present experiments only at the end of the different contractions when the muscles were just at the point of fatigue. Consequently, blood flows at this time presumably most accurately reflect the increasing intramuscular pressure due to the increasing tension exerted. The actual blood flows at the end of the contractions could be measured, in many experiments, but for some of the conditions examined it was necessary to extrapolate from the results shown in Fig. 1. The results for all three subjects with forearms immersed in water at 34° C are shown in Fig. 3.

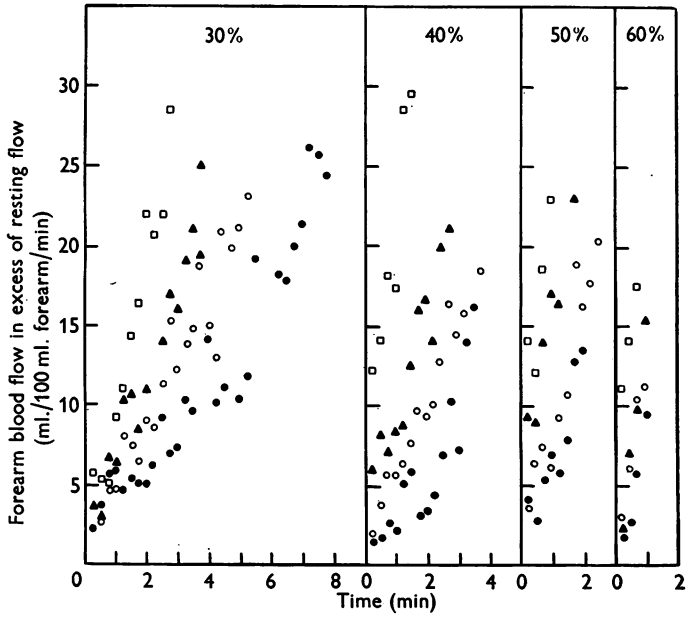

Fig. 2. Forearm blood flows, in excess of resting flows, for subject A during contractions of 30, 40, 50 and 60 % of maximal voluntary contraction, with the forearm immersed in a water-bath at either 18° C (●), 26° C (○), 34° C (▲), or 42° C (□).

In general, as the tension increased, the final blood flows decreased, and if the curves shown in Fig. 3 are extrapolated, they suggest that blood flow through the forearm would be occluded when the tension exerted was above some value greater than 70 % MVC. This view is supported by the few flows that could be measured at a tension of 70 % MVC, which were all between 2 and 11 ml./100 ml./min higher than the resting values.

The post-exercise hyperaemia followed the same pattern as has been reported before (Clarke *et al.* 1958), increasing approximately linearly with

the duration of the contraction for any given tension; as expected, for a given duration of contraction the post-exercise hyperaemia also increased with tension.

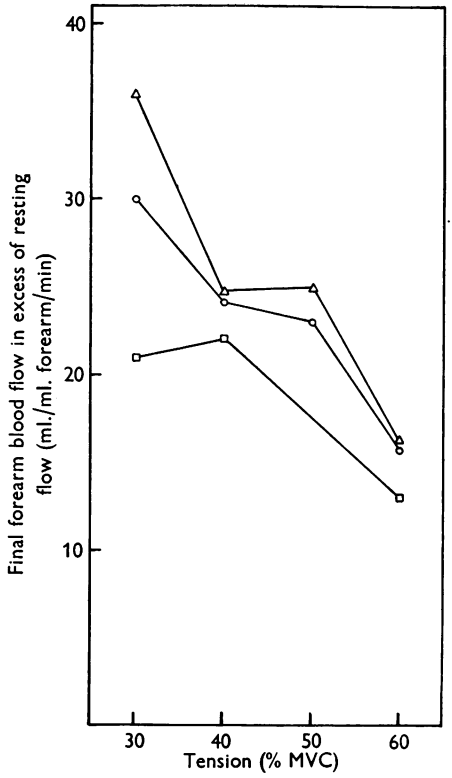

Fig. 3. The forearm blood flows, in excess of resting flows, for three subjects at the end of contractions sustained to fatigue at tensions of 30, 40, 50 and 60 % of maximal voluntary contraction. The forearms were immersed in a water-bath at 34° C. Some of the values were obtained by extrapolation from Fig. 1. △ subject P; ○ subject A; □ subject K.

Blood-flow measurements through the whole forearm might reflect changes either in the active muscles or in the muscles that were not active in the contraction, or even, conceivably, in the skin. The evidence from plethysmographic recordings from the whole arm is therefore, by itself, not conclusive. To try to define more precisely where in the forearm the observed changes in blood flow occur, an electromyographic study was performed to determine (1) which muscles participated strongly in the hand-grip contractions, and (2) which muscles were completely inactive. Following this, temperature changes in both active and inactive muscles were measured after the forearm had been pre-heated or pre-cooled.

Repetition of these experiments with artificial arterial occlusion to the contracting arm allowed the deduction of changes of blood flow through the individual muscles.

Electromyographic evidence

From the electromyographic study of the muscles in the forearm, three muscles were found which played a large part in the hand-grip contractions at all tensions. They were (1) extensor carpi radialis, (2) flexor digitorum sublimis, and (3) flexor digitorum profundis; of these, the first two played the largest role in this type of contraction, as judged by the amount of electromyographic activity recorded. No activity whatsoever was detected in the flexor carpi radialis or the flexor carpi ulnaris during these hand-grip contractions.

Muscle temperature measurements

Temperatures were recorded simultaneously in several forearm muscles at a time, usually in two of the active muscles and in the two inactive muscles mentioned above. The temperature of the muscles was reduced by immersion of the forearm in well stirred water at 18 or 26° C; changes in muscle temperature during and after contractions in cool water are illustrated in Figs. 4, 5 and 6. Figure 4 shows typical results from one subject with his arm immersed in water at 26° C. during contractions at 30 % MVC. In the first contraction the circulation was free and during the contraction there was a rise of temperature of 4° C in both the flexor digitorum sublimis and the extensor carpi radialis (i.e. active muscles) while in the flexor carpi ulnaris and the flexor carpi radialis (inactive muscles) the rise in temperature was only about 0·5° C. Twenty minutes later the subject repeated the contraction, but with the circulation arrested: on this occasion the temperature did not rise in any of the four muscles during the contraction. Immediately after each contraction (the circulation was restored at the end of the second contraction), the temperature of the active muscles increased rapidly to reach a peak within 2 min of the end of the contraction, thereafter falling more slowly to a steady level. The inactive muscles showed a much smaller and slower rise of temperature, which reached a peak some 2–5 min after that found in the active muscles.

Figure 5 shows essentially similar results from another subject also performing a contraction of 30 % MVC, but in water at 18° C. Furthermore, in this experiment the circulation was arrested during the first contraction and was free in the second contraction. In the first contraction the temperature of the active muscles increased slightly, but much less than during

the second contraction, while the temperature of the inactive muscles did not change on either occasion.

Regrettably, this technique is not suitable to follow temperature changes in muscles during contractions at high tensions, when the force of the contracting muscles not infrequently results in movement of the needles. Furthermore, as can be seen in Fig. 4 and 5, temperatures changes took some time to occur and at the higher tensions the contractions were too

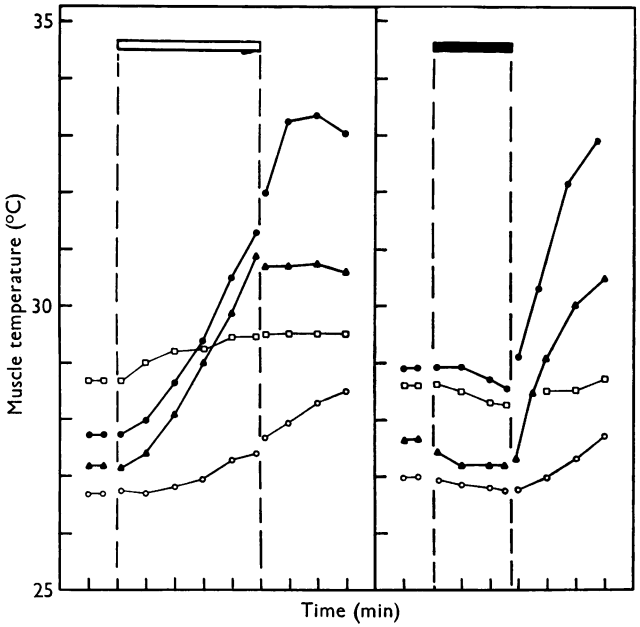

Fig. 4. Muscle temperatures during a sustained contraction ▭ of the forearm at 30 % maximal after pre-cooling in a water bath at 26° C. On the right is shown another contraction 20 min later, at the same tension, with arterial occlusion of the arm ▬, ▲ Flexor digitorum sublimis, ● extensor carpi radialis brevis, □ flexor carpi radialis, ○ flexor carpi ulnaris. Subject P.

short for their development. Nevertheless, consistent changes in muscle temperature were found during contractions up to 50 % MVC, for which tension Fig. 6 shows results obtained from several experiments in a water-bath at 26° C. As before, during contractions both with and without free circulation there was no change in the temperatures of inactive muscles. The active muscles at this tension characteristically showed a small rise in temperature when the circulation was occluded, but a much greater rate of rise when the circulation was free (2·5–4 times greater in the flexor digitorum profundis and the flexor digitorum sublimis, while in the extensor carpi radialis the increase in the rate of rise of the temperature was 1–1·5 times greater).

Figure 7 shows the temperature changes found when contractions of 30% MVC were performed, with and without artificial arrest of the circulation, after the temperature of the muscles had been substantially raised by previous immersion of the forearm in water at 44° C. During the first contraction, when the circulation was free, the temperature of the active muscles fell while that of the inactive muscles remained unchanged.

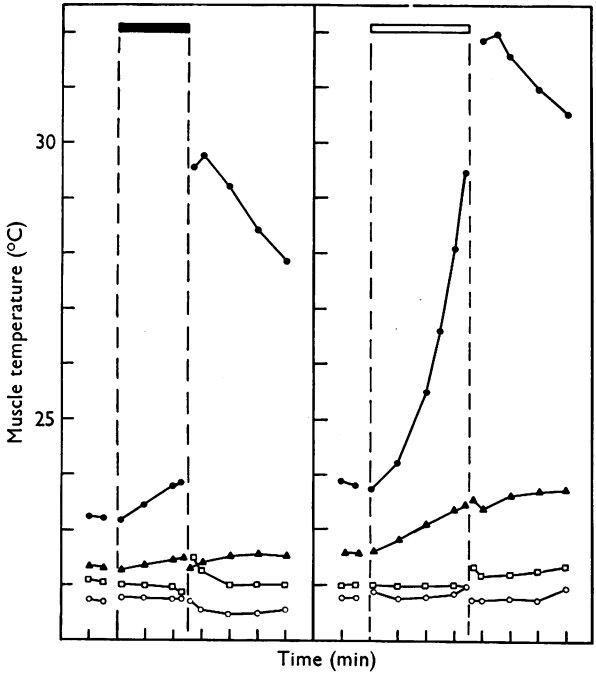

Fig. 5. Muscle temperatures during sustained contraction of the forearm at 30% maximal after pre-cooling in a water-bath at 18° C. On the left with arterial occlusion of the arm ■, on the right 20 min later without occlusion □. Other conventions as in Fig. 4. Subject R.

When the contraction was repeated with circulatory arrest the temperature of both active and inactive muscles increased steadily throughout the period of occlusion. After the contractions ended the temperature of the active muscles fell steeply to a trough within a few minutes of the end of the contraction, while the inactive muscles showed a small and slower reduction of temperature.

Blood-pressure recordings

Blood pressure was recorded by auscultation on the opposite arm of the subject before, during and after many contractions. Characteristically, whatever the tension, there was a rise in both systolic and diastolic pressures by 10–20 mm Hg within 10–15 sec of the start of the contraction.

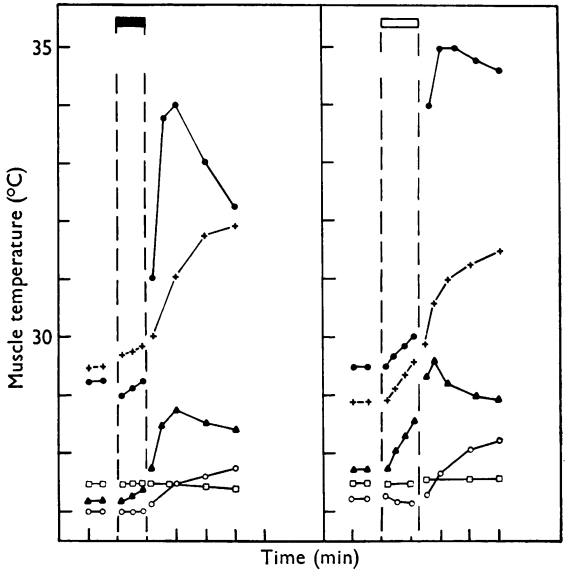

Fig. 6. A composite result of three experiments on one subject where muscle temperatures were measured during sustained contractions of 50 % maximal after pre-cooling in a water-bath at 26° C; on the left with arterial occlusion of the arm ■, on the right 20 min later without occlusion □. + Flexor digitorum profundis. Other conventions as in Fig. 4. Subject P.

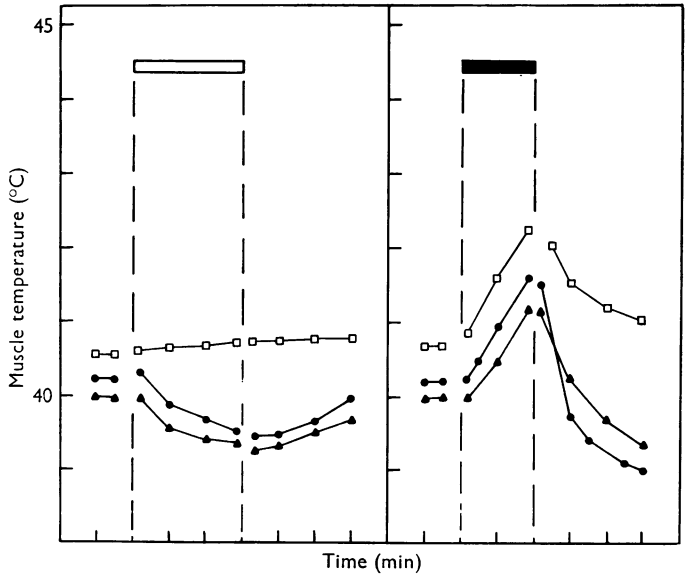

Fig. 7. Muscle temperatures during sustained contraction □ of the forearm at 30 % maximal after pre-warming in a water-bath at 44° C. On the right is shown another contraction 20 min later with arterial occlusion of the arm ■. Other conventions as in Fig. 4. Subject P.

Thereafter both pressures rose throughout the contractions at a rate dependent on the tension exerted, until at the point of fatigue the systolic pressure reached values of 170–190 mm Hg while diastolic pressures reached 150–170 mm Hg. After the contractions ended both systolic and diastolic pressures fell rapidly, to reach resting values in all cases within a few minutes. These changes are illustrated in Fig. 8, which shows typical responses from one subject during a contraction at 30 % MVC. No attempt has been made in these experiments to elucidate the cause of these changes,

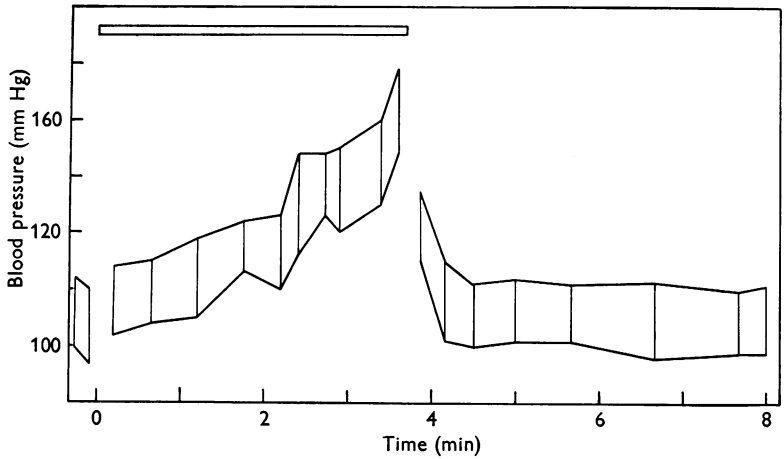

Fig. 8. Blood pressures measured by auscultation, before, during and after a sustained contraction at a tension of 30 % of maximal voluntary contraction at a water-bath temperature of 34° C. Subject A.

especially since the auscultatory method of recording blood pressure is not regarded as a highly accurate one. To ensure that the rise of blood pressure was not due to pain, some measurements were made at a tension of 10 % MVC. At this low tension contractions can be maintained almost indefinitely, and do not produce pain. Yet both systolic and diastolic pressures rose by some 10–20 mm Hg soon after the contractions started and never fell below this level for as long as the experiment lasted (the longest duration was for 1 hr).

Duration of contractions with and without artificial occlusion

The functional significance of the blood flow to the active muscles of the forearm is demonstrated in Fig. 9, which shows, for two subjects, the durations of sustained contractions at different tensions when the arm was immersed in water at 34° C. The open circles represent the durations of contractions when the circulation was not artificially impeded, while the solid triangles represent the durations when artificial arterial occlusion was

applied to the upper arm. The two curves appear to converge as tension increased, but even at tensions of 60 % MVC and 70 % MVC application of artificial occlusion to the contracting arm reduced the duration of contraction by 10 % or more.

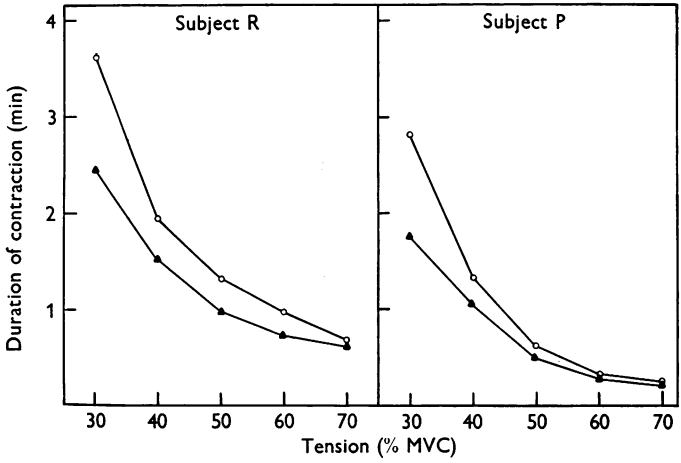

Fig. 9. The durations of contractions sustained to fatigue for two subjects at tensions from 30 to 70 % of maximal voluntary contraction, with the forearm circulation free (○) and artificially occluded (▲). The forearms were immersed in a water-bath at 34° C.

DISCUSSION

The present experiments show clearly that the blood flow through the forearm increases during hand-grip contractions sustained at tensions up to at least 70 % MVC. The blood flow through the forearm increased steadily throughout these contractions and may amount to as much as 40 ml./100 ml./min over the resting values. The evidence from direct measurement of the blood flow through the whole forearm itself suggests that the increased flow is confined to or occurs largely in the active muscles. Thus, the vessels in the skin and in the inactive muscles are less likely to account for the increased flow for one or other of the following reasons: (1) The pattern of response was similar at all water-bath temperatures from 18 to 42° C. (2) There was a progressive reduction of the final blood flows through the forearm at the occurrence of fatigue as tension increased (see Fig. 3); this may reasonably be interpreted as a reduction of blood flow through the active muscles due to a steady increase of intramuscular pressure, particularly since at the point of fatigue the blood pressures were similar in all contractions.

Conclusive evidence is supplied by the measurements of temperature changes in active and in inactive muscles during contractions with and

without artificial occlusion of the circulation after pre-cooling or pre-heating the muscles in order to exaggerate the difference in temperature between the muscle and the incoming arterial blood; any increase in the amount of blood to the muscle would be expected to result in a change of muscle temperature towards that of the blood. In its present form, this technique has two principal shortcomings. First, as already mentioned, it is not possible to obtain reliable information at high tensions, and secondly, the technique will not admit of quantitative analysis. Despite these drawbacks results obtained from all tensions up to 50 % MVC showed consistently, (i) a change in the temperature of active muscles towards the temperature of the incoming arterial blood during contractions when the circulation was free, a change that could be abolished or markedly reduced by artificial arterial occlusion of the arm, and (ii) little or no change in the temperature of inactive muscles towards that of the arterial blood temperature, whether or not the circulation was occluded. The inescapable conclusion from the present experiments is that the increase of blood flow during sustained contractions of the forearm, measured by the Whitney strain-gauge plethysmograph, goes preponderantly, if not entirely, to the muscles active in the contraction.

This conclusion is, perhaps, somewhat surprising, in view of the demonstration (Fig. 8) that both systolic and diastolic pressures increase considerably in response to sustained contractions of a few muscles in the forearm even when the tension they exert is low. While there are earlier reports of large rises of blood pressure during sustained contractions involving large muscle groups (e.g. Monod, 1956), it has not been suspected that such large rises could result from the weak contractions of only a few muscles in the forearm. This increase in blood pressure may well have a functional importance in maintaining the supply of blood to the contracting muscles to overcome the intramuscular compression of the vessels. The evidence suggests that there is little or no increase in the blood flow to the inactive muscles or to the skin; in view of the large increase in blood pressure found here during contractions, and taking into account the arterial dilatation that is claimed to occur as a result of local muscular activity (Hilton, 1959), it is possible that there is vasoconstriction in the inactive muscles.

The present finding that blood flow to contracting muscle is not occluded until the tension exceeds some value in excess of 70 % MVC is supported by the fact that artificial occlusion decreased the duration of the contractions at all tensions examined. This conclusion conflicts with the findings of Barcroft & Millen (1939), who showed that the blood flow through the calf is occluded during contractions with tensions of less than 20 % MVC. While the size of the muscles in the forearm and the calf, and the leverage systems involved in the two limbs, are quite different and may presumably

account for some of the discrepancy noted in the critical tension at which the blood supply is occluded, it is difficult to see how this discrepancy, of more than 50 % MVC, can be explained in this way. The difference may be partly explained by the reportedly higher intramuscular pressure during contractions in which the muscle shortens than in isometric contractions (Mazella, 1954). The most likely explanation for the occlusion of the blood flow to the calf in Barcroft & Millen's experiments at a tension of less than 20 % MVC was put forward originally by Barcroft & Swan (1953), that in the calf the blood vessels were 'nipped' as a result of the massive shortening of the soleus and gastrocnemius muscles. In the present experiments, with nearly isometric contractions, this presumably did not occur, so that there was only the intramuscular pressure to compress the blood vessels.

During the post-exercise period the hyperaemia does not involve the skin vessels (e.g. Grant & Pearson, 1938). During the post-contraction hyperaemia, however, changes of temperature were recorded both in active and in inactive muscles. Irrespective of whether or not artificial occlusion was applied during the contraction, there was a sharp change in the temperature of the active muscles towards blood temperature, whereas in the inactive muscle there was a smaller and much slower change of temperature, with a time course which lagged some 5 min behind that in the active muscles. It is presumed that the change of temperature in the active muscles can only be due to an increased blood flow. The change of temperature of the inactive muscles in the post-contraction period might also be due to an increase in blood flow, in which case the flow is presumably much smaller in extent than that in the active muscles, and is delayed in its onset; alternatively, it is suggested that the change of temperature of the inactive muscles could be accounted for, or at least greatly influenced, by heat conducted from adjacent active muscles: such a hypothesis would explain not only the smaller change in temperature but also the delay in the start of the change. It seems likely, then, that just as has been deduced during the contraction itself, the increased blood flow through the forearm after the contraction is entirely or primarily through the muscles active in the contraction.

It is still uncertain how the hyperaemic response to exercise is evoked or controlled. It has been suggested, from experiments on anaesthetized animals (Hilton, 1959) that spinal (or higher) reflex activity does not take part in the hyperaemia due to exercise. This belief may possibly have to be reconsidered in view of the results from the present experiments under physiological conditions, in which both the systemic and local cardiovascular responses, occurring promptly with the onset of contraction, are suggestive of the co-ordinated activity commonly associated with nervous reflex control.

SUMMARY

1. Forearm blood flow measured by plethysmograph during hand-grip contractions sustained to the point of fatigue showed an increase in the amount of blood flowing to the forearm at tensions from 30 to 60 % MVC. The blood flow increased steadily during each contraction, to reach values at the end of the contractions of from 10 to 40 ml./100 ml./min greater than the resting blood flow level.

2. The rate of rise of blood flow during the contractions increased with tension, but the actual values at the end of the contractions decreased as tension rose. The results obtained suggest that intramuscular pressure during contraction cannot occlude the blood supply until the tension exerted is above some value greater than 70 % MVC.

3. Specific evidence on the destination of the increased blood flow was obtained from an electromyographic investigation supported by the measurement of temperature changes in both highly active and totally inactive muscles of the forearm during the hand-grip contractions. The increased blood flow went preponderantly, if not entirely to the muscles active in these contractions.

4. Surprisingly large increases in blood pressure were recorded, even during relatively weak contractions. The implications of this finding, in view of the evidence obtained on blood flow through the forearm, on the haemodynamic responses to muscular exercise are discussed.

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